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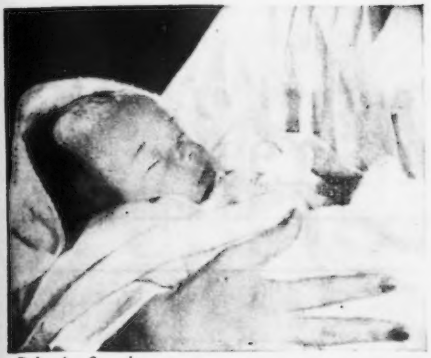
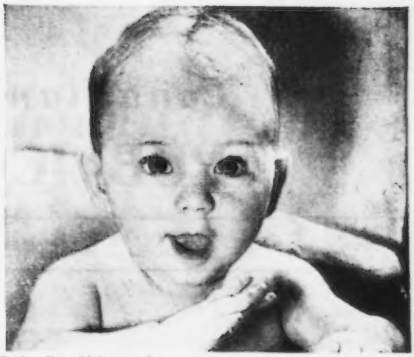


Baby A—2 weeks



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The Epidemiology and Pathogenesis of Type E and Fish-Borne Botulism

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THE known isolations of *Cl. botulinum* type E, as summarized in earlier papers[1, 2] must now be extended to include seven additional instances which have come to light within the past three years. The main purposes of this communication are to bring the record of type E botulism up to date, to amplify previous reports of two strains recently isolated in our own laboratory, and to suggest possible explanations of the conspicuous role of fish as vehicles for this type of botulism.

Total Known Isolations of Type E Strains

The salient data bearing on all known isolations of type E strains are set forth in the table. Of the five instances listed here for the first time in the English literature, two occurred in Russia, one in France, one in Japan, and one in Denmark. These occurrences will be described below, along with some expansion of earlier references to the beluga and trout strains, isolated by us in 1950 and 1952 respectively.[3]

U.S.S.R. In a rare monograph on the pathogenesis of botulism, published in Moscow in 1949, Matveyev[4] states that a type E strain was isolated by Kushnir in 1937 from a red-fish caught in the Sea of Azov. In 1940, Zavadovskaya[5] referred to this isolation as having taken place in 1934. Unfortunately, neither Kushnir's original account,[6] nor the author herself, is accessible for consultation on this question; but it seems improbable that the earlier year is correct. The available evidence bearing on the history of the prototype strains[7] indicates that three cultures, supposedly "of a new toxicological variety",

were despatched in December, 1935, by Dr. L. Bier, Director, Bacteriological Institute, Dniepropetrovsk, U.S.S.R., to Dr. K. F. Meyer, Director, George Williams Hooper Foundation, University of California, San Franscisco, U.S.A. When the cultures were received in March, 1936, one of the tubes had broken in transit, and no culture could be recovered from it. The two surviving cultures were promptly and definitely shown to represent a new type by Gunnison, Cummings and Meyer[8], who proposed for it the designation *Clostridium botulinum*, type E. In August, 1936, four additional cultures (including a replacement of the one lost in the earlier consignment) were forwarded to Dr. Meyer by Dr. E. Kushnir of the above-mentioned Institute, and these arrived in San Francisco in March, 1937. The belief of the Russian workers that these belonged "to the same variety" as the preceding cultures was soon confirmed. Five of the six strains had been isolated from the

KNOWN ISOLATIONS OF CLOSTRIDIUM BOTULINUM TYPE E

Year	Reported by	Cases	Deaths	Place of Occurrence	Source of Culture
1935–36?	Gunnison, Cummings and Meyer (1936)	—	—	Ukraine, U.S.S.R.	Sturgeon intestines (5)and muscle(1)
1934	Hazen (1937)	3	1	Westchester County N.Y., U.S.A.	German commercially canned sprats
1934	Hazen (1938)	3	1	Cooperstown, N.Y., U.S.A.	Labrador smoked salmon
1937?	Kushnir (1937)	—	—	Sea of Azov, U.S.S.R.	Red-fish
1938	Zavadovskaya (1940)	1	1	Leningrad, U.S.S.R.	Smoked herring
1941	#Geiger (1941)	3	1	San Francisco, Calif., U.S.A.	Yugoslav mushrooms canned in California
1944	Dolman and Kerr (1947)	3	3	Nanaimo, B.C., Canada	Home-canned local salmon (also home-canned chicken and soil of chicken run)
1949	Dolman, Chang, Kerr and Shearer (1950)	2	1	Vancouver, B.C., Canada	Home-pickled local herring (also jejunal contents of fatal case)
1950	#Meyer and Eddie (1951) Dolman and Chang (1951)	5	0	Point Hope, Alaska, U.S.A.	Uncooked beluga (white whale) flippers
1951	Prévot and Huet (1951)	—	—	North-central France	Fresh-water perch intestinal contents
1951	Nakamura, Iida and Saeki (1952)	14	4	Iwanai, Hokkaido, Japan	Vinegared herring and rice cakes
1951	Pedersen and Freundt**	6	0	Denmark	Pickled Herring
1952	Dolman (1953)	1	1	Natal, B.C., Canada	Home-pickled trout from local river

#Culture not isolated. Type E toxin identified.
**Personal communication from Dr. Inga Scheibel, State Serum Institute, Copenhagen.

intestines of sturgeon (*Acipenseridae*) and the other from the muscle "of a kind of sturgeon".

These historical comments are intended to establish that the Russian literature contains no reference to type E strains before publication of Gunnison, Cummings and Meyer's report; and are provoked by Zavadovskaya's failure, four years later, to mention their important contribution. It is readily conceded that the first type E strains were isolated in the U.S.S.R., and that they were recognized there as possibly representing a new type—albeit mainly in the light of negative toxin-neutralization tests with samples of types A, B, C, and D antitoxic sera forwarded to them from the University of California—but this does not entitle Soviet scientific literature to overlook the important contribution of these United States workers in establishing the identity and fundamental characteristics of *Cl. botulinum*, type E.

Zavadovskaya herself reports the isolation of a type E strain from smoked herring, which had caused a single fatality from botulism in Leningrad, late in 1938. A man ate a large amount of the fish, and developed the typical symptomatology of botulism 16 hours later. He died about 45 hours after the onset of illness, despite the administration of polyvalent types A and B botulinus antitoxin. The identity of the culture was established through a sample of type E antitoxin procured from the Bacteriological Institute at Dniepropetrovsk, and the author asserts that hers is apparently the first account of human botulism due to a type E strain. But the two episodes investigated in 1934 in New York State by Hazen[9, 10] obviously take precedence over the Leningrad fatality. Zavadovskaya alludes to Hazen's 1937 report, but does not mention her second (perhaps inaccessible) paper, published in 1938, in which the cultures isolated from both these outbreaks were specifically identified as type E. Incidentally, Zavadovskaya's claim does indicate that the original sturgeon strains, contrary to our previous supposition[2], were not implicated in outbreaks of human botulism. They were doubtless isolated in the course of microflora surveys, designed to throw light on the mechanism of fish-borne botulism, of which the contemporary Russian literature records several examples.[11, 12, 13]

France. Early in March, 1951, in northern France, a family of three developed botulism after consuming pickled pilchards. This episode, which was not bacteriologically investigated, focussed the attention of Prévot and Huet[14] on type E botulism, and led them to survey the intestinal contents of a large number of fish. They examined 163 fresh-water fish (representing 8 common species) from two ponds, located within 100 miles of Paris; and 13 salt-water fish (11 mackerel and 2 pilchards), caught in the English Channel. Of these 176 fish, 11 yielded cultures toxic for mice, and from one of them, a fresh-water perch (*Perca fluviatilis*), a strain of *Cl. botulinum* type E was isolated. A 5-day filtrate of the perch strain had a toxicity of 100 mouse M.L.D. per ml., neutralizable by rabbit antitoxic serum prepared from the Vancouver herring ("VH") strain, a sample of which had been sent by us to Dr. Prévot at the Pasteur Institute, Paris. In our own laboratory, a subculture of the perch strain has yielded toxins having a potency of 1000 mouse M.L.D. per ml. This type E strain from France resembles the "VH" strain in

many respects, including the capacity of fresh culture filtrates (in amounts equivalent to 5,000 mouse M.L.D.) to induce paralysis on subcutaneous injection into young chickens.

Alaska. Meyer and Eddie[15] have already reported on an outbreak of five cases of mild botulism among Alaskan natives at Point Hope, Alaska, on the Chutkchi Sea, in 1950. The food responsible was beluga flippers (forelimbs of the white whale, *Delphinapterus leucas*), prepared by storing the partially dried but uncooked flippers in seal oil for several months in the owner's hut. In that locality, this much-esteemed delicacy is eaten raw on special occasions. Meyer and Eddie demonstrated the presence of small amounts (about 4 mouse M.L.D. per gram) of type E toxin in a sample of the suspected material, but failed to isolate *Cl. botulinum* therefrom. However, as previously reported[16, 3], from a similar sample kindly sent us in November, 1950, by Dr. W. H. Gaub, Arctic Health Research Center, Anchorage, Alaska, we managed to isolate a micro-organism having the colonial, biochemical and toxigenic characteristics of *Cl. botulinum,* type E. Cultures grown in ground meat beef infusion medium for 3-5 days at 37°C., yielded toxic filtrates of about 1000 mouse M.L.D. per ml., which were neutralized by antitoxic sera prepared from rabbits inoculated with toxoids of the Nanaimo[1] and Vancouver[2] type E strains. This beluga strain resembles the Nanaimo cultures in that their toxins fail to cause paralysis when injected into chicks, in amounts equivalent to 20,000 mouse M.L.D.

Japan. Late in May and early in June, 1951, at a village named Shimano, near Iwanai, on the island of Hokkaido in northern Japan, 14 local inhabitants developed symptoms of botulism after eating herring "*izushi*"—cakes made of vinegared herring and rice. Four persons died, the first victim being a 59-year-old woman who made the cakes. A few days after her death, a gathering of neighbours and friends at her home partook of the remaining fish cakes, following which there were three other fatalities, involving a woman aged 37, a man of 54, and a boy of 16. Two women, aged 41 and 27, and a young man of 20, were seriously ill, but recovered. All these individuals, who had eaten a considerable amount of the cakes, showed many of the classical features of botulism—vomiting, stomach pains and bloating, constipation, hoarse voice, impaired vision, difficulty in breathing, thirst, and normal or sub-normal temperature. Several of the patients also displayed drooping of the lids, dilated pupils, and diplopia. The remaining 7 milder cases comprised 3 adults and 4 children, all of whom had eaten only a slice or two of the herring "*izushi*". Their chief complaints were vomiting, stomach-ache, diarrhoea, thirst, and general tiredness.

This outbreak is doubly interesting as the first recognized occurrence of botulism in Japan, and as the largest type E outbreak yet recorded. The responsible food, very popular in the district, was prepared according to custom by mixing steamed rice, carrot and rice-yeast with sliced raw herring, this mixture being preserved in vinegar and spices, and pressed in a tightly closed container for about 3 weeks, when it was eaten cold. From the incriminated foodstuff, Nakamura, Iida and Saeki[17] isolated a strain of *Cl. botulinus* type E, which they identified through type cultures sent to Dr. Yuko Tohyama, Chief,

Food Control Division, National Institute of Health, Tokyo, by Dr. K. F. Meyer of San Francisco, who kindly drew our attention to this outbreak. Through the courtesy of Dr. Tohyama, we received a subculture of this Japanese strain, and have verified its type E characteristics.

Denmark. Through the kindness of Dr. Inga Scheibel, State Serum Institute, Copenhagen, we learned of another type E outbreak after this communication first went to press. Few details are available of the hitherto unreported episode, which occurred in Denmark in 1951. There were 6 cases, none of them fatal, and the botulinic vehicle was pickled herring. A strain of *Cl. botulinum* isolated from the fish was eventually identified as type E by Drs. H. O. Pedersen and E. A. Freundt.

British Columbia. Towards the end of September, 1952, a middle-aged man residing at Natal, a small community in the southeast corner of British Columbia, developed signs and symptoms of botulism, after consuming home-pickled trout. The patient died in hospital, after the notably long lapse of 18 days. Clinical and epidemiological data relating to this episode are already on record[3], as is the fact that we isolated a type E strain from a companion jar of the trout cutlets. To discourage premature abandonment of quests for type E organisms in suspected material, a few details may relevantly be given here respecting the isolation of this latest strain in the series of four which have now come our way.

Since the jar of fish received by us was tightly sealed, with contents of normal appearance and odour, while neither the pickling fluid nor a saline extract of the fish were toxic for mice, prospects seemed unpromising. But on the assumption that endogenous spores (present in the gut of the fish when caught) might have vegetated and migrated to form toxin in the muscles under appropriate post-mortem conditions, a thorough search for *Cl. botulinum* was instituted. Generous amounts (2-4 gms.) of the fish were inoculated into a 1% peptone-beef infusion medium containing ground meat, which previously had been boiled and cooled to around 90°C., with 2% glucose then added. No aerobic growth resulted, but after 3-4 days of incubation at 37°C. in a McIntosh-Fildes jar, marked gas production was evident, without apparent proteolysis of the meat particles. Stained preparations showed Gram-positive bacilli of rather inconstant size, some bearing an oval, sub-terminal spore. Well-dried, freshly prepared human blood agar plates, streaked with a large loopful of this fluid culture, and then incubated at 37°C. under complete anaerobiosis for 2-3 days, showed a moderate number of slightly haemolytic, greyish-white, ovoid colonies, 1-2 mm. in diameter, with smooth edges and a finely granular texture. On plates streaked while moist, growth was arborescent or confluent. A few non-haemolytic colonies of another sporulating bacillus also developed, but caused little confusion in this instance.

The carbohydrate-fermenting activities were determined, using bromthymol-blue as indicator, on cultures grown for 48 hours at 37°C. in a McIntosh-Fildes jar, the medium being beef infusion, to which 1 per cent of Difco peptone and 1 per cent of the test sugar had been added. The reactions corresponded, in the main, with those shown by other type E strains, viz., acid production *positive* for glucose, fructose, sucrose, maltose and sorbitol; and *negative* for lactose, arabinose, xylose, inulin, mannitol, dulcitol, and starch. Gas production, under the conditions specified, was slight and variable. Other features of the trout strain consistent with a type E identity were the hollow and occasionally moth-eaten appearance of the bacilli, and their irresolute Gram-positivity; and the heavy white growth deposit which formed among the meat particles in the medium, contrasting with the slight turbidity of the supernatant fluid. Further, suspensions of the trout strain were agglutinated to high titre by a rabbit antiserum prepared against one of the prototype Russian strains.

Despite these hopeful features, culture filtrates were non-toxic for mice. The episode was almost set aside as unproven, when a culture held at room temperature (23°C.) after

several daily transfers in the ground meat medium containing 2% glucose, began to produce gas vigorously and showed other evidence of good growth. The supernatant contained about 1000 mouse M.L.D. per ml., neutralized by type E antitoxin. The potency of toxin subsequently produced in this medium at 23°C. or at 37°C. ranged from 300-1000 mouse M.L.D. per ml. Thus the trout strain resembled other type E strains in its comparatively low toxigenic capacity, as judged by lethal effects on mice; and also in its ability to produce toxin at temperatures well below 37°C. In the absence of glucose, the trout strain grew feebly, and produced very little toxin at either 23°C. or 37°C.

Summary. Recorded isolations of *Cl. botulinum* type E, as summarized in the table, now total 20. Assuming that the six cultures originally sent from Russia were distinct strains, but that the multiple isolations in the Nanaimo and Vancouver episodes represented homologous cultures, then 17 separate type E strains are known to have been identified since 1935—a rate of one per annum. Only three of these strains (those isolated by Kushnir and Zavadovskaya, and the recently identified Danish strain) have not been available to us for study. The main cultural, biochemical, and serological properties of this group of 14 type E strains may be detailed in future publications. Meanwhile, the following fundamental data relating to type E botulism call for interpretative comment. Ten outbreaks have now been reported, involving 41 persons, of whom 13 died. Four of these outbreaks occurred in the United States (including one in Alaska), three in Canada, one in the U.S.S.R., one in Japan, and one in Denmark. Eight were fish-borne, while in one a marine mammal was the vehicle. In addition, type E strains have been isolated from presumably normal fish on seven occasions in Russia, and once in France. These facts, which perhaps reflect the ecology of the causal micro-organisms, will be discussed under the headings fatality rate, geographic distribution, and predilection for fish.

Fatality Rate

Among 1,281 cases of botulism reported in the United States[18] during the period 1899-1949, the fatality rate was 65 per cent. The corresponding rate for 56 cases recognized in Canada between 1919 and 1952 was 57 per cent[3]. In Europe, by contrast, the case fatality rate has been much lower. For instance, Wasmuth[19] quotes an overall rate of 15 per cent for Germany; while in France during the occupation the mortality in over 1,000 cases was less than 2 per cent[20]. Obviously, the case-fatality rate in a given country will be affected by the extent to which milder attacks of the disease are recognized; but these divergencies in prevailing rates on the two continents are too wide to be imputed entirely to different degrees of diagnostic acumen.

A more plausible explanation would appear to be that the types of *Cl. botulinum* implicated in a given area greatly influence the local case-fatality rate. Although less than one-third of 487 North American outbreaks have been bacteriologically identified, type A strains have clearly preponderated, the reported isolations of types A, B and E cultures totalling 121, 26, and 6, respectively. In Europe, on the other hand, human botulism has been chiefly due to type B organisms. The tendency for type A strains in general to display higher toxigenic capacities (as judged by mouse lethality) than type B strains, may at first seem to favour the above hypothesis. But of course the lethal

effects of botulinus toxins injected parenterally into mice do not necessarily parallel their potentialities when taken orally by man. Many type C-alpha strains, for example, produce toxins whose mouse-killing power is a thousand-fold higher than that of the average type E toxin; yet to date the former have never been identified with human botulism, whereas type E intoxication can be fatal for man in less than 24 hours.[1]

A quarter-century ago, Meyer[21] emphasized that botulinus toxin production was greatly influenced by the nature of the foodstuff implicated. More recently, Legroux, Levaditi and Jéramec[20] noted during the upsurge of botulism in occupied France that the gravity of an attack apparently did not depend upon the *type* of toxin ingested, and that fatalities were more apt to be associated with watery comestibles, such as conserved stews, jellied meats, and vegetables. A foodstuff's botulogenic propensity may be affected not only by the aqueous content, but also by its protein and carbohydrate composition; by its pH, consistency and homogeneity; and even by its shape and bulk. In fact, dissimilarities in the foodstuffs customarily responsible for botulism in various parts of the world (processed meats, especially hams, sausages, and other pork products in Europe, home-preserved vegetables and fruits in the United States, and fish in the Soviet Union), may be major determinants of local case-fatality rates.

The limited statistics available on fish-borne and on type E botulism are not inconsistent with the last-mentioned hypothesis. According to Matveyev,[4] among 609 cases of "fish-poisoning" recorded in Russia during the pre-revolutionary era, 1818-1913, there were 283 deaths, a fatality rate of 45.5 per cent. Although a large majority of these poisonings were no doubt botulinic—the syndrome had been recognized in that country for more than a century—they entirely lacked bacteriological confirmation; for the first reference in the Russian literature to *Cl. botulinum* isolated from fish dates from 1914.[22] On the grounds that erratic reporting would tend to stress the more disastrous episodes, it seems probable that the actual case-fatality rate for fish-borne botulism in Russia has been somewhat lower than the above calculated figure. This deduction finds support in later figures, compiled in 1937, and cited by Zavadovskaya.[5] Among a series of 168 cases of fish-borne botulism in the Soviet Union, there were 56 deaths, a fatality rate of 33 per cent. Incidentally, the mortality was much higher among persons whose illness was traced to small, netted fish (smoked or salted herring, bream, carp, roach or perch), than in botulism due to red-fish (various types of sturgeon and large salmon). Of 23 cases in the former category, 16, or 70 per cent, died, whereas of 145 cases in the latter category, only 40 died, i.e. 27 per cent. Such a difference can hardly be ascribed to heterogeneity of vehicle, but could be due to slower diffusion and more irregular distribution of toxin in the larger fish.

Although Zavadovskaya's figures are probably more accurate than those culled from pre-revolutionary records, they are again unsupported by bacteriological information; and since her series antedates recognition of type E, the proportion of outbreaks due to these strains would be conjectural anyhow. Nevertheless, the quoted case-fatality rates for fish-borne botulism in Russia

are strikingly similar to the 31 per cent rate calculated from the data in the table. This proximity, while consistent with an epidemiological relationship between fish-borne and type E botulism, does not discountenance the contention that lethality in human botulism is determined more by the nature of the foodstuff than by the type of *Cl. botulinum* implicated. A case-fatality rate of around 30 per cent, intermediate between North American and European figures, therefore may express chiefly the botulogenic propensities of raw or insufficiently cooked fish. On the other hand, the wide range (from nil to 100 per cent) in the type E episode mortality rate, may derive from the peculiar vagaries and labilities of type E bacilli, spores, and toxins.

Geographic Distribution

Of the 10 type E outbreaks now reported in accessible literature, 7 have occurred in North America. There are three plausible explanations of this, each of which will be considered briefly:

i. *That North American bacteriologists have been particularly alert for type E episodes.*

To uphold this thesis without giving offense would require considerable finesse, and it is fortunate that so delicate an operation need not be attempted here. The prepared mind and previous acquaintanceship are of course helpful in the recognition of *Cl. botulinum* type E, but no extremely tricky techniques or complex media are entailed.* The failure of bacteriologists in many countries to encounter this micro-organism can be explained adequately by postulating a relative dearth of type E spores in the regional soils and waters. The complete lack of reported isolations so far from the southern hemisphere, which is especially noteworthy, can hardly be attributed to prevailing technical inadequacies, or to unawareness of the relevant literature. After all, *Cl. botulinum* types C-beta and D were described first in Australia and South Africa respectively.

ii. *That North American diets predispose to type E botulism.*

In the United States, of 477 botulism outbreaks summarized by Meyer and Eddie,[18] only 27 (under 6 per cent) were attributed to fish and sea foods.[2] In Canada, on the other hand, 6 out of 10 known outbreaks have been due to fish or a marine mammal.[3] Although two of the Canadian outbreaks involved natives in the Far North, who tend to be heavy consumers of uncooked fish and marine products, a ten-fold disparity in the incidence of fish-borne botulism cannot be explained solely in terms of dietary differences between these countries. The inhabitants of North America as a whole eat comparatively little fish. By contrast, despite the popularity of fish foods in e.g. many parts of the United Kingdom and Germany, no type E botulism has been reported from these countries; and only one such outbreak is known to have occurred in Japan, where fish—often lightly cooked, pickled, or raw—is a dietary staple. Hence, the extent to which fish features in the community diet is apparently not the major determinant of the geographic distribution of type E botulism.

*Until supplies of specific antitoxic sera are available, suspected type E cultures may be forwarded to these laboratories for confirmatory tests.

iii. *That type E spores are especially prevalent in North American soil and littoral waters.*

Bacteriological surveys of soil samples, together with the reported incidence of human and animal botulism in various parts of the world, suggest that spores of the longer-known types of *Cl. botulinum* are irregularly scattered. Admittedly, wrong conclusions may be drawn from inadequate surveys, inaccurate diagnosis and reporting, and statistical fallacies. For instance, it was inferred erroneously for many years that type C-beta spores occurred only in Australia, and type D only in South Africa. Again, although the uneven distribution of types A and B spores is established, the complete absence of either type from a given area should not be assumed. Thus, Nakamura *et al.*[17] relate the apparent non-occurrence of botulism in Japan (before the recent outbreak near Iwanai) to the presumed lack of indigenous *Cl. botulinum* spores; but they have to qualify their argument by adducing several hitherto unpublished isolations of type A strains from Japanese soil. Incidentally, in that country, botulism might easily be mistaken for the more familiar tetrodon poisoning, of which hundreds of cases develop annually, with a fatality rate of from 30 to 50 per cent.[23]

Notwithstanding such reservations, the following statements bearing on type E spore distribution seem valid. First, 4 out of 10 proven type E outbreaks have occurred in the north-western fringe of this continent, and were due to salmon, herring, white whale and river trout caught in that general area. Secondly, although one of the 3 remaining North American outbreaks was traced to Labrador salmon, the other two were attributed to sprats from Germany, and to mushrooms originating in Jugoslavia. Thirdly, type E strains have actually been isolated on the European and Asiatic continents—from a fresh-water perch in France, from pickled herring in Denmark, from normal sturgeon and botulogenic herring in the Soviet Union, and from a herring-containing foodstuff in Japan. These facts appear to fit the hypothesis that type E spores are widespread, at least in the northern hemisphere, with especially heavy disseminations in the soils and waters of certain parts of North America, and probably of the U.S.S.R.

Predilection for Fish

The evidence pointing to the special liability of type E botulism to be fish-borne needs no recapitulation. Before any attempt is made to elucidate this association, it should be emphasized that the relationship does not necessarily hold in reverse. In other words, all fish-borne botulism should not be regarded automatically as of type E origin. For example, it is tempting to speculate that in addition to the 3 British Columbian type E outbreaks, 2 other occurrences, viz. the Indian fatalities in the Yukon due to salmon eggs,[24] and the Eskimo disaster from seal meat in the North West Territories,[3] may have resembled bacteriologically as well as epidemiologically the beluga-flipper cases among the Alaskan Eskimos. In that event, one-half the recorded outbreaks of botulism in Canada could be classed as both of type E origin and of piscine or marine mammalian conveyance—a remarkably prevalent con-

junction, the more so since the corresponding proportion for the United States is roughly 0.6 per cent. But this form of argument remains speculative, even when bolstered by further quasi-specific evidence such as that offered by Prévot and his associates.[25, 26] They reported recently from France two separate cases of botulism due to pickled tuna, affecting men aged 18 and 48 years, whose ocular and other paralyses persisted without amelioration until repeated doses of type E toxoid had been administered. Although in neither instance was a type E culture isolated, nor any toxin demonstrated, the authors imply that fish-borne and type E botulism may be regarded as synonymous. Reciprocal logic of this type should be spurned not merely because it ignores several contravening facts, but also because it could retard clarification of the factors underlying the pre-eminent role of fish as vehicles for type E botulism.

Bacteriological verification that botulism could be fish-borne was first recorded in 1907 by Madsen,[27] who briefly alluded to isolating *Cl. botulinum* from toxic mackerel—presumably caught in Danish waters. According to Meyer,[7] this was a type B strain. In 1948, Wasmuth[19] described a series of 30 cases of type A botulism in north-western Germany, due to vinegared herring. During the intervening 40 years, in the United States, 3 fish-borne outbreaks were identified as due to type A organisms, and 2 to type B;[18] while in the Soviet Union, these types were similarly implicated on numerous occasions. Although type A outbreaks predominated in the U.S.S.R., the liability of either type to be involved was recognized in the manufacture of both A and B antitoxic sera for therapeutic purposes. For example, Kolesnikova *et al.*[28] reported in 1937 a typical severe outbreak affecting 33 persons who had eaten sandwiches containing smoked sturgeon. Of 6 victims given no antitoxin all died; whereas only 2 died among the remaining 27 cases (including 16 very ill), all of whom received type A antitoxin or a mixture of A and B antitoxins.

The Russian literature of the decade preceding the isolation of the prototype E cultures contains many indications of official concern over the high incidence of botulinic fish poisoning, which resulted in extensive enquiries into the sources, mechanisms, and prophylaxis of fish pollution with *Cl. botulinum*. These investigations included laboratory experiments on the pathogenesis of fish-borne botulism;[4, 29] bacteriological surveys of the intestinal contents and external wounds of fish caught in the Caspian Sea, Mahach-Kala Lake, and other waters;[11, 12] and a search for *Cl. botulinum* in soil, water, sea slime, and other possible contaminating agents, sampled in the vicinity of fish salting factories.[13] Much additional information bearing on these questions is furnished in a series of mimeographed reports sent in September, 1936, to Dr. K. F. Meyer at the University of California by Prof. V. Bronner, Director, Foreign Sanitary Information Bureau, Moscow.[30]

The conclusions reached by these workers must be briefly summarized. Ruchkovsky[29] first suggested, in 1928, that fish may become botulogenic through diffusion of toxin elaborated by intestinal organisms, which proliferate and migrate into the tissues of the fish after its death. He showed that sturgeon-type fish were insusceptible to enormous doses (250,000 guinea-pig

M.L.D.) of type A toxin administered intragastrically, although smaller species of fish, e.g. herring, carp and minnow, died with typical botulinic symptoms from relatively little toxin. Further, he demonstrated the development of toxicity in the muscle of salted and dried sturgeon which had been inoculated intragastrically, before being sacrificed, with large numbers of toxin-free type A organisms. In these laboratories, before Ruchkovsky's work had come to our attention, similar results (unpublished) were obtained, *mutatis mutandis*, by transferring gold-fish to water containing toxin-free type E organisms.

The contention that fish might thus serve as 'healthy carriers' of potentially botulogenic spores received ample confirmation from subsequent surveys of their intestinal contents. For instance, of 56 suspicious, but non-toxigenic cultures isolated from 182 red-fish (varieties of sturgeon) by Burova and Nasledischeva,[12] 17 agglutinated with a type A antiserum. Again, Kibalchich, Komkova and Muromcev[30] isolated type A or B strains from the intestines in 7 of 130 (5.4 per cent) red-fish, and in 7 of 103 (6.8 per cent) netted fish—mostly carp, bream and catfish—caught in the northern Caspian Sea, the chief fish-producing region of the Soviet Union. Many more positive samples (15 per cent) were secured from fish long dead than from those examined immediately after death (2.4 per cent). It was concluded that spores in the water are ingested by live fish; that once caught, fish should be promptly eviscerated and kept cold; and that the prevalent practice of salting small, ungutted fish in large barrels was particularly dangerous. The same authors demonstrated that in red-fish, external wounds and bruises, and internal wounds caused by baited iron hooks, might also serve as portals of entry for spores. They isolated *Cl. botulinum* types A or B from wounds on 6 out of 50, or 12 per cent, of sturgeon caught in the Astrakhan region in 1934. Matveyev[4] cites additional evidence to the same effect. One survey, conducted in 1936-38, and involving 1,258 red-fish samples, yielded 63 (5 per cent) type A, and 14 (1.1 per cent) type B cultures. Also isolated were 11 cultures having typical characteristics of *Cl. botulinum*, but whose toxins were not neutralized by polyvalent A and B antitoxin. The evidently unchecked possibility that some of this last group may have been type E cultures betrays a limited awareness of the existence of this type in the U.S.S.R. at that time.

The foregoing references abundantly confirm that fish-borne botulism is not necessarily due to type E organisms. Also, the previously unavailable Russian literature substantiates the importance of both endogenous and exogenous routes of infection in the development by fish of types A or B botulogenic propensities. These pathogenetic alternatives are equally applicable to type E organisms, as we contended (largely on *a priori* grounds) when reporting the Vancouver herring episode. But the apparent predilection of type E strains for fish is not explained thereby. More light is thrown on this question by other suggestions advanced in earlier papers,[1, 2] for which there is now much supporting evidence, and which therefore merit amplification.

The isolation of type E strains from improperly canned chicken and from the soil of a near-by chicken-run, and the detection of type E toxin in canned mushrooms, render their affinity for fish non-exclusive. Yet if their spores be

as widely distributed as the accumulated data now indicate, the rarity of non-piscine type E occurrences seems explicable only in terms of such postulated properties as low heat resistance, specialized environmental circumstances for toxin production, and marked capacity to survive in fresh and salt water. Experimental results, to be published elsewhere in due course, validate each of these postulates, of which the first-mentioned seems the most fundamental. All workers with type E cultures have commented on the unusually slight heat-stability of their spores. In these laboratories, the susceptibility of different strains in this respect has been observed to fall within a rather wide range. For instance, while the spores of certain strains occasionally withstood exposure to 100°C. for as long as 30 minutes, other specimens failed to germinate after only 5 minutes' exposure to similar conditions. Such observations could largely account for both the paucity of type E outbreaks involving heat-processed foodstuffs, and the apparent predilection of type E strains for fish, which are sometimes eaten pickled, smoked, or otherwise 'preserved', but yet uncooked. Even should spores survive, germinate, and produce toxin in a foodstuff, the toxin itself is apt to be particularly heat-labile, so that slight exposure to heat has a prophylactic effect. Toxin of the "VH" strain, for instance, almost completely degenerates in a few days at 37°C. Finally, when outbreaks of type E botulism do occur, failure to reckon with heat-lability may result in negative bacteriological findings, thus again contributing to under-estimation of the extent and density of distribution of this type of *Cl. botulinum*.

Although most workers in this field have noted the fickle toxigenic activities of type E cultures, the only relevant point on which there seems general agreement is that many of these strains have an optimal temperature for toxin production around 30°C. Good growth and toxin production may, however, proceed at room temperature (23°C.), while small amounts of toxin may develop even at 6°C.[2] This finding, in view of the special liability of fish to be contaminated with *Cl. botulinum* spores (which of course may survive indefinitely under cold storage conditions) seems to accentuate the botulogenic hazards of uncooked fish. Ingram's references to the limitations underlying the chilling and freezing of foodstuffs, have special pertinence to this situation.[31] Again, although the association of type E outbreaks with pickled fish at a pH around 5.0 or less[2, 3] does not necessarily point to toxin being produced at that degree of acidity,[32] our laboratory experience suggests that unusually low pH values are compatible with type E toxin production. Yet another factor warranting mention here is the chemical composition of fish muscle protein, which might be singularly propitious for the elaboration of type E toxins. In this laboratory, it was observed that a glucose-peptone-beef infusion medium containing added *cooked* fish, in place of the customary ground meat, yielded weak type E toxins. On the other hand, several type E strains produced toxins of much higher potencies in pulped *raw* fish than in mashed raw meat. One type A strain was tested, and did equally well in either of these media.

Finally, there remains the question of spore survival in water. In 1935, Burova *et al.*[13] isolated 3 toxigenic type A strains from 150 soil samples,

2 from 69 sea water samples, and 1 from 49 samples of sea slime, collected in the vicinity of fish factories on the Sea of Azov. Six samples of river water, and 6 of river slime, were negative. A search for type E strains in surveys of comparable scope would be a formidable task, in view of the frequent presence in soil and water of many spore-bearing organisms of high thermal stability. But it has been shown in these laboratories that type E spores, inoculated into tap and sea water, proved as durable as types A and B spores, and more so than type C spores, under similar experimental conditions. Spores of 4 type E strains, as well as of one type A and one type B strain, showed some survivors after 10 weeks in sterile samples of sea water, held at either 6° or 23°C. Such spores, when prevalent in soil bordering on rivers, lakes or sea, would have a fair chance of being ultimately ingested—perhaps at long distances from their telluric starting-point—by fish or a marine mammal. The ecology of type E spores may include occasional vegetation and multiplication within the gut of these vectors, so that the spore content of heavily polluted inland waters, rich in big fish (as in parts of the Soviet Union), is replenished by excretion from their intestines. Some proliferation of these anaerobes might also occur intermittently in sludge or slime at the bottoms of lakes and rivers, or in the ocean depths.

Summary

1. Since 1934, there have been 10 known episodes of proven type E botulism, involving 41 persons, of whom 13 died.
2. Seven occurrences were in North America, including 3 in British Columbia, 2 in New York State, one in California, and one in Alaska. One case was reported from Russia in 1940, and a 14-person outbreak from northern Japan in 1951. An outbreak in Denmark, also in 1951, has just come to light.
3. Some form of fish, or a marine mammal, was implicated in all but one of these outbreaks. *Cl. botulinum* type E has been isolated also from the intestinal contents of sturgeon in the Soviet Union, and of a river perch in France.
4. In Russia, where fish-borne botulism has been prevalent for more than 100 years, both exogenous and endogenous modes of infection of fish with *Cl. botulinum* type A or B have been demonstrated. The intestinal tract of large sturgeon-type fish is particularly prone to harbour such organisms. A similar pathogenesis probably obtains in type E botulism.
5. Although fish-borne botulism is by no means invariably of type E origin, fish have undoubtedly served as the customary vehicles of type E botulism. This association seems mainly due to the slight heat resistance of type E spores, which tends to prevent their survival in even moderately heated foodstuffs. Other possible contributory factors are that type E toxin is likewise markedly heat-labile, and is produced at unusually low temperature and pH ranges. Also, fish protein may be particularly conducive to toxin production by type E strains.

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Infantile Gastro-enteritis Associated with Escherichia Coli

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SO much literature has been published about the etiology of infantile gastro-enteritis that it would not be possible to summarize it here. Kauffmann and Dupont's[6] publications, as well as those of Modica,[10] give a good review of the problem.

The belief that certain serological types of *Escherichia coli* could be the causative agents of epidemic infantile gastro-enteritis is now commonly accepted. British investigators, in 1948, first isolated strains of *E. coli* with particular antigenic characteristics which they found almost invariably in gastro-enteritis epidemics. These strains were described under different names: *Bacterium coli neapolitanum*,[1] *Bacterium coli* α and β,[4] *B. coli* B.G.T.[15] or *B. coli* D 433.[18] Kauffmann has studied and classified these strains and today we know three types, called *E. coli* 111/B4, *E. coli* 55/B5, and *E. coli* 26/B6.[14]

Research is now being carried forward in France. P. Nicolle[12,13] has developed a method of bacteriophage typing which is of great epidemiological interest.

In the United States, two interesting studies concerning this subject were published in 1950[11] and 1952.[10] We do not know of anything yet published in Canada relating to studies on *E. coli* 11/B4, 55/B5 or 26/B6.

We have had the opportunity of doing research in this particular field and the results of our work are presented in this paper.

Agglutinating sera and specific strains were kindly supplied by the Pasteur Institute, in Paris,* and we were able to begin our research at the Hôtel-Dieu St. Vallier, in Chicoutimi, on July 15, 1952. From that date on, we examined stools of all infants admitted to the hospital with a diagnosis of gastro-enteritis.

We have not had an epidemic in the hospital, but we have had to deal with sporadic cases from the region. The information received from the few localities from which the most serious cases came, indicated that real epidemics

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existed. With the cooperation of the Health Department,* investigations were made in the families where we found babies who had a positive reaction to *E. coli* sera. The staff of the nursery also was examined, but none was affected. One hundred and ninety specimens were examined; this number does not include the repeat examinations for each patient. Of these specimens, 127 were from babies not older than 15 months.

Age Distribution. Some authors have pointed out that *E. coli* organisms are found in infants under one year; other investigators maintain that *E. coli* is more frequent in infants from a few days' old up to seven months, or from birth to three months. Our cases were among babies from 18 days to 15 months, most of the positive cases being from 18 days to 6 months. These children were artificially fed. British authors consider artificial feeding as a condition favouring the disease, not because of contamination of the milk by *E. coli* organisms, but because of the general condition of the baby and its nutritional problems.

Bacteriology. As we have said, stool specimens were taken in every case of gastro-enteritis occurring among babies. The following technique was used. Specimens were streaked on to the surface of S.S. agar, blood agar and Endo agar plates. We searched for Salmonella and Shigella as well as for *E. coli.* With blood agar medium we found it easier to obtain a pure culture of *E. coli,* to study its characteristics, and to notice the association of other bacteria. Those most frequently found were *Streptococcus faecalis, Staphylococcus aureus,* and sometimes *B. proteus.* This can be explained by the fact that our patients did not come to the hospital at the onset of their illness. On blood agar medium we more often obtained pure cultures, which were very characteristic.

Of the 36 positive cases of gastro-enteritis associated with *E. coli,* 24 had auricular complications of otitis media and mastoiditis. The bacteriological examination of auricular pus very often showed the presence of *Staphylococcus aureus,* but *E. coli* was never found. In one case we isolated *Klebsiella ozenae.*

The suspected colonies were first tested for agglutination with specific sera by the slide method. Then the test tube method was performed with living agar cultures, and heated broth cultures. Biochemical study and I M V C reactions were also made for each isolated strain.

From the routine cultures already mentioned, we isolated 36 pathogenic *E. coli,* 32 being type 55/B5 and 4 type 111/B4. We did not isolate the third type described by Orskov, type 26/B6.

We also noticed the particular seminal odor, mentioned by the British authors, in all the strains of *E. coli* 111/B4.

Our strains were sent to the Pasteur Institute, Paris, where the serological type was confirmed by the Salmonella Service. The strains were then given to the Bacteriophage Service** for phage typing. Our *E. coli* strains proved to be

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of type "Sèvres III" and "Sèvres I" for the 111/B4 strains, and type "Lomme II", "Graz", and "Bethune" for the 55/B5.

Epidemiology

We have no particular comments to make, as far as epidemiology is concerned, apart from those usually made with any contagious disease transmitted through the digestive tract. It is certain that these specific diarrhoeas are contagious. We had an opportunity to confirm this in the family of baby G.G. and F.G. (nos. 7 and 13 in the table). We also observed this fact in the hospital, where, in spite of the greatest care, we had a few instances of infection, especially of the air-borne type. In fact, naso-pharyngeal swabs from many patients of our department have shown the presence of the same organisms found in the faeces.

This could be explained in two different ways: air contamination or vomiting, but we could not establish their relative importance. We may affirm with certainty that we found the coliform organism in the nasal secretions of three babies not affected by vomiting. Their stools at that time were free of the specific bacteria. A few days later we finally isolated from their stools type 55/B5. This had previously been found in their nasal secretions (see cases nos. 18, 20 and 21 in the table).

One may conclude, therefore, that transmission is not only by infected hands; we must take into account also that infection can be air-borne.

We cannot be certain about the length of the incubation period since we have had but a few cases of infection in the hospital. We share the opinion of R. Buttiaux,[2] who says that it is usually 5 to 10 days in length.

Discussion and Conclusion

It is becoming increasingly obvious that *E. coli* is the cause of certain gastro-enteritis epidemics in infants. Many arguments favour this hypothesis. In our series of 127 cases, the bacteriological findings are in agreement with the clinical facts. We had 4 serious cases (all fatal) in which the pathogenic micro-organisms were not detected. But if we must consider the possibility of a virus in the case of negative cultures, we must also think of the impossibility of obtaining control cultures and the possibility of errors in technique. Two of our cases died 24 hours after entering hospital, making it impossible for us to obtain a second stool culture.

In a few cases (nos. 9, 17, 18) we had to take 3 rectal swabs before detecting the pathogenic micro-organism. Although it has been said by a few authors that pure cultures of *E. coli* are found at the beginning of the disease, we do not think that this can be considered a rule. In the cases mentioned above, we obtained a pure culture only after 10 or 14 days. At the onset of illness we found mixed cultures of *Streptococcus faecalis* and *Staphylococcus aureus* and that type of *E. coli* that is found in normal faeces. This was due, perhaps, to the fact that the antigenic characteristics of *E. coli* were not yet changed.

In support of the hypothesis of the etiological role of *E. coli* in these cases

	No.	Name	Age	Adm. date	Cl. asp	Complic.	Stool cult.	N. pha cult.	Sero. aggl.	Evol.	Epidemiology phage typing
	1	Ro.M.	2m	25.7	mild	otit. med.	31.8			recov.	Lomme II
	2	Tr.B.	1m	9.8	mild	otit. med.	31.8			recov.	Lomme II
	3	Gl.S.	4m	22.8	mild	otit. med.	31.8			recov.	Lomme II
	4	Bo.P.	4m	31.8	severe	otit. med. antrotom.	9.8			recov.	Lomme II
	5	Sl.O.	6m	4.9	toxic		5.9			died 4.9.	Lomme II
	6	Sh.D.	1m	12.9	moder.		13.9			recov.	Lomme II
	7	Ga.G.	2m	25.8, 12.9	severe	otit. med. antrotom.	13.9	13.9		relap.	Lomme II
	8	Du.B.	4m	1.9	moder.	otit. med. antrotom.	26.9			recov.	Lomme II contact No. 4
	9	Du.P.	7m	29.8, 7.9, 11.10	moder.	otit. med.	26.9		1/80	relap.	Lomme II contact No. 4
	10	Br.C.	4m	17.9, 1.10	mild		2.10			relap.	Lomme II contact No. 7
	11	Ga.S.	1m	11.9	severe	otit. med. antrotom.	6.10	6.10		recov.	Lomme II
	12	Sa.A.	4m	24.9	moder.	otit. med.	7.10			recov.	Lomme II
	13	Ga.F.	5m	4.10	severe	pyoderm.	7.10	..		recov.	Lomme II cousin to No. 7
	14	Po.G.	3m	6.10	mild		7.10			recov.	Graz
	15	Tr.M.	4m	25.9	moder.	otit. med.	12.10	..		recov.	Lomme II
	16	Tr.J.	3m	14.10	toxic	otit. med. antrotom.	15.10	..		recov.	Lomme II
Esch. coli 55/B5	17	Be.C.	1m	17.10	severe	otit. med. antrotom.	23.10		1/80	recov.	Bethune
	18	Bo.R.	40d	25.9, 14.11	moder.	otit. med. antrotom.	28.10	24.10		relap.	Lomme II contact No. 11
	19	Tr.D.	4m	30.10	toxic	otit. med.	1.11		1/160	recov.	
	20	Ga.M.	6m	18.10	mild	eczema otit. med.	3.11	28.10	1/80	recov.	contact No. 17 16.21
	21	Pa.R.	6m	16.10, 1.11	mild	otit. cong.	9.11	7.11	1/80	relap.	contact No. 18 20
	22	Co.G.	3m	11.11	moder.	otit. cong.	12.11			recov.	
	23	Fo.R.	18d	13.11	mild	pyoderm.	18.11	..		recov.	
	24	SG.L.	6m	15.10, 6.11	moder.	otit. med. antrotom.	22.11	..	1/160	relap.	
	25	Ga.F.	2m	22.10, 22.11	mild	otit. med.	23.11	..	1/40	relap.	
	26	Th.G.	4m	21.11	mild	eczema otit. med.	10.12	8.12		recov.	contact No. 25 27

	No.	Name	Age	Adm. date	Cl. asp	Complic.	Stool cult.	N. pha cult.	Sero. aggl.	Evol.	Epidemiolog phage typing
	27	X.El.	2m	7.11	moder.	pyoderm.	10.12	..		recov.	contact No.26, 25
	28	Ga.F.	21d	14.12	toxic	bronch. cong.	17.12	..		died	
	29	Be.H.	5m	18.12	toxic	otit. med.	19.12	..	1/80	recov.	
	30	Sa.R.	15m	18.12	mild	otit. med.	19.12	..	1/320	recov.	
	31	St.D.	18d	26.11	moder.	pyoderm.	20.12	..		recov.	
	32	Vi.R.	6m	26.12	moder.		27.12	..		recov.	
	33	Si.L.	10m	22.8	toxic		23.8			died 23.8	Sevres III
Esch. coli 111/B4	34	Tr.C.	3m	3.10	mild	otit. med.	4.10			recov.	Sevres I
	35	Bl.G.	1m	19.11	severe	otit. med.	20.11		1/801	recov.	
	36	Du.F.	15m	12.11	moder.	rachit.	13.12	..	1/80	recov.	

Cl. asp.: clinical aspects; Evol.: evolution; Complic.: complications;
Stool cult.: positive culture of the stools. N. pha. cult.: positive culture of the naso-pharynx.

of diarrhoea, we might consider the three babies mentioned above (cases nos. 18, 20 and 21). Since these babies were not affected by vomiting, and *E. coli* was isolated from their nasal secretions and only later from the faeces, this would indicate that the infection was air-borne and due to their proximity to other patients.

We do not think it necessary to consider the association of any virus with *E. coli* to explain the diarrhoea that we studied. We must rather admit that there are gastro-enteric conditions due to food only, in which cultures remain negative, but in which pathogenic organisms can develop. Illness due to improper food, as well as other disease, can favour the transformation of *E. coli*. It is admitted that pathogenic *E. coli* of type 111/B4 or 55/B5 was seen more often among babies artificially fed. On the other hand, *E. coli* 111/B4 was rarely found among adults, and very seldom in older children. Does a general organic deficiency favour an increase of virulence, or an eventual mutation with a transformation of the antigenic structure of existing *E. coli*, in a given environment? These are problems to be solved.

It has been said, in refuting the etiological role of *E. coli* in infantile gastro-enteritis, that no agglutinins are found in the patient's blood. This statement is losing more advocates every day. Already a few authors such as Gilles,[4] Modica,[10] and Julien Marie, have found specific agglutinins. We also obtained positive agglutination by means of heated suspensions, in the sera of 10 patients, in dilutions of 1/40 to 1/320 (see table). Increasing numbers of positive agglutinations will be found as our techniques improve. It seems to us that the production of agglutinins is likely earlier in some infants than in

others. The particular reaction of each individual against an organism such as *E. coli,* which is a normal inhabitant of the intestinal tract, and the age of the patient, contribute to a large extent in explaining the differences in the production of antibodies.

Thirty-two babies were found to harbor *E. coli* 55/B5 and 4 babies *E. coli* 111/B4. All these cases were quite severe. Recovery required from 10 to 21 days. The treatment given was a large dosage of antibiotics and a plentiful supply of fluids, as well as transfusions of whole blood. No other pathogenic organism has been detected as the cause of the illness. We searched for agglutinins in the serum of 10 patients who remained longer in the hospital, and found specific agglutinins. During convalescence, control cultures from faeces showed that the pathogenic coli gradually diminish and then completely disappear. Streptomycin and chloromycetin were used as bactericidal agents and seemed to give good results.

In conclusion, the specific phage typing of the *E. coli* strains and the consistent pattern of both bacteriological and serological findings, leads us to affirm that some of the gastro-enteric conditions found in infants are caused by *E. coli* of specific antigenic structure.

Summary

The authors describe 36 sporadic cases of infantile gastro-enteritis, in which they detected *E. coli* 55/B5 and 111/B4 as specific organisms.

No other organisms were detected which could have explained these serious cases of diarrhoea.

Hospital infection and the presence of the same organism in the rhino-pharynx of the patients confirm the hypothesis as to the epidemic character of these cases of gastro-enteritis, the etiological role of *E. coli,* and the possibility of air contamination.

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Letter from Great Britain

Social Stratification and Disease

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Dear Editor,

THE search for health in the Western World has led research workers in recent years increasingly to study the incidence of disease, and the factors which are associated with it, in relation to the circumstances which surround the individual in his life in the community. For the people of Britain the decennial census has provided an exceptional opportunity to further this work, and since we are now in process of studying the Census of 1951 it is a convenient moment to consider what is the significance to be attached to the Registrar General's attempts to group people into categories and to study the varying mortality picture presented by them. It must be said at once that the mortality studies based upon the 1951 Census have not yet been completed. But it may be of value to recall previous studies mainly devoted to the previous census of 1931.

One of the most interesting studies is that which defines people by social class. This term, in the sense used in mortality and morbidity analyses, is based upon occupation. The assumption is that occupations, at which man spends most of his working hours, must stamp those engaged in them in a uniform pattern of life. Man's associates at work will, in fact, mould him.

Moreover it is a striking fact that occupations can be arranged in an order of 'prestige' which conforms closely to the average of public opinion. This is, of course, true only in the broadest sense since there cannot possibly be precise agreement in the details of all occupations. As Professor Glass has said, 'Farmers and farm labourers may agree on the social rating which each group affords the other. But such agreement is far less likely to be reached by, for example, farmers and artists.'

The Registrar General groups the six-hundred-odd occupational groups of Britain into five social classes which in substance date back to T. H. C. Stevenson's mortality tables of 1911. The nature of these classes will be understood by the broad descriptions: (1) professional etc. occupations; (2) intermediate occupations; (3) skilled occupations; (4) partly skilled occupations; (5) unskilled occupations.

Social class 1, comprising 2.4% of the total (545,000), consists in the main

of those who have undergone an extensive training probably at university level: professional engineering, surveying and architecture (144,000), the medical profession (58,000), the legal profession (26,000), scientists, (50,000), clergy (48,000), officers of the Armed Forces (46,000), literature (29,000), directors of business (144,000). At the other end of the scale, social class 5, comprising 11.8% of the total (2,669,000), consists of those engaged in occupations which carry no responsibility and a minimum of skill: labourers (just under two million), cleaners (half a million), and other lowly occupations such as costermongers, watchmen, newspaper sellers, and porters (200,000).

The largest group, comprising 52% of the total (11,750,000), is class 3, typified by the skilled artisan. This consists of clerical workers (2,005,000), shop assistants (1,146,000), personal service (421,000), foremen (565,000), and skilled craftsmen (7,614,000).

Class 2, comprising 14.8% (3,341,000), contains a varied assortment of persons with responsibility over the lives of others: managing directors and employers of business (492,000), various professional persons and officials (408,000), teachers (357,000), clerks engaged in costing, estimating and accountancy (490,000), hotel and restaurant keepers (235,000), farmers (338,000), medical auxiliaries (263,000), proprietors and managers of wholesale and retail businesses (758,000).

Finally, class 4, comprising 18.9% (4,278,000) contains the semi-skilled worker. This group includes, for example, the agricultural worker, the locomotive fireman as opposed to the driver, and the bus conductor as opposed to the bus driver.

Studies based upon this classification depend upon the accuracy of the census returns and upon the care taken, when registering births and deaths, in determining the occupation, i.e. the precise nature of the work done by each person and of the trade or industry with which it is connected. As the functions of compiling and registering are conducted by different persons, there is the possibility that the Census computations and the local registrar's records of occupation will not precisely coincide. To this inaccuracy must be added those already mentioned associated with the groupings themselves, and the fundamental errors of diagnosis. Little study of this problem has yet been made but that already done shows mortality and morbidity variation within the social classes that are sufficiently striking to call for a careful study. The facts are, of course, well known and are summarized briefly as follows.

The most striking mortality differences within the five social classes are to be found in infant mortality. Stillbirth rates, neonatal rates and postnatal rates all rise steeply from class 1 to class 5. The differences are greatest for babies surviving to beyond the first month. The gradient for deaths of infants certified as due to infection is very marked. Moreover, it is most striking that the marked differences which can also be shown for infant mortality rates in different parts of Britain are not present for babies in social class 1. There is no appreciable difference for deaths due either to congenital or birth injuries, but a sharp gradient from 1 to 5 can be seen for deaths due to prematurity. For deaths due to cancer of the uterus the gradient rises from social class 1 to social class 5; for deaths due to cancer of the

breast the rates rise in the opposite direction. These social class differences are present both in married and unmarried women. Death rates from cardiovascular changes as indicated by coronary thrombosis, and those for appendicitis, influenza, diabetes and syphilis decline from class 1 to class 5. For duodenal ulceration there appears to be no marked differences between one class and another, while deaths from gastric ulcer rise steeply from class 1 to class 5.

Here, then, we have evidence of many differences in habits of life. In a picture of vast complications certain obvious causes of mortality variations are detectable. Thus, we can distinguish variations in opportunities for infection, greatest in the overcrowded households of the unskilled workers and least in the homes of the professional classes, differences in food consumption, in exercise, in occupational risk; distinctions in the sexual pattern of life and in the advantages of education, all of which will reflect themselves in many ways adverse to class 5, while the increases in mental stress and strain rise from class 5 to 1. We can do no more than guess at the nature of much that is happening.

All this is of the greatest significance to public health. We look forward in this country to the detailed studies of the 1951 Census which may do much to increase our knowledge of this important branch of social pathology.

A Diphtheria-Carrier Study amongst Navy and Army Personnel

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FOR SOME TIME public health experts in Canada and the United States have been concerned that, because of a general decline in the incidence of diphtheria, the immunity of the general population has not been maintained through natural exposure to this disease.[1] This low level of immunity is particularly evident in adult groups who do not regularly receive booster doses of diphtheria toxoid. This has been demonstrated by Greenberg and Fleming,[2] as well as others, who reported that a high percentage of adults in their series demonstrated no measurable antitoxin before immunization.

Because of this changing situation, epidemiologists and immunologists have considered it advisable to keep watch on the diphtheria-carrier rate in general and selected populations.[3, 4]

Organization

The opportunity was afforded the writer, while on special duty with the Royal Canadian Navy, to conduct a diphtheria-carrier survey amongst naval personnel at Esquimalt, British Columbia, and army personnel at Gordon Head and Work Point, British Columbia. The advice of members of the Department of National Health and Welfare, and the School of Hygiene and Connaught Medical Research Laboratories, University of Toronto, was sought and given. The naval service expressed willingness to co-operate in any way possible.

The survey was conducted at the naval base at Esquimalt, where approximately 2,000 personnel were based. As laboratory facilities to handle such a large volume of work were not available within the Naval Service, arrangements were made with the Central Laboratory of the Department of Health of Ontario to examine the swabs. It was agreed that 500 nose and throat swabs per week over a period of four weeks would be shipped air express to Toronto. In addition, it was possible to arrange with the director of the laboratory of the Department of Veterans Affairs Hospital in Victoria, B.C., for the examination of 97 specimens.

Methods

Final details of the survey were arranged with the Command Medical Officer for the West Coast, and it was decided to set up a mobile team to

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carry out the survey. This team was to proceed from place to place, meeting the personnel to be examined rather than requiring the personnel to come to meet them. As most of the personnel at H.M.C.S. "Naden" and "Dock Yard" were taking courses in various schools, it was a simple matter to arrange for the schools to be dismissed for the period of time required to examine the men.

The nose and throat swabs were obtained by a petty officer with considerable experience in sick bays, both on ships at sea and at bases on shore, and by the writer. A typist prepared a list of names and assigned a code number to each person's name. The number was then transferred to a completed questionnaire form which was filled out by each man being examined. The fourth member of the team wrote the individual's name and code number on each tube.

Sterile tubes and swabs, prepared by the central supply room of the hospital, consisted of two applicators made into swabs and inserted into a plugged tube which was autoclaved from one-half to three-quarters of an hour at 15 pounds' pressure. On such short notice it was impossible to obtain regular-size test tubes in quantity. However, the medical stores had on hand a considerable quantity of presumptive Kahn tubes, and after checking with other laboratories it was decided that these would be satisfactory.

Procedure

Before the survey was done at any one place, a short explanatory talk was given the personnel to be examined, after which they received a slip on which they wrote their name, age, official number, rating or rank, length of time served in their present location, name of their previous place of residence and the length of time served or lived there as a civilian. A "remarks section" was reserved for noting whether or not the person had ever had diphtheria or been a diphtheria carrier; if so, the approximate date was given. All persons who were examined had previously been given toxoid or, if in the Service for more than one year, had received a booster dose annually. The completed form was given to the clerk preparing the list and he copied down the man's name, official number, and rank, and assigned a code number to his slip. The code numbers ran from 1 to 2,093. The men to be examined then proceeded to one of the two persons carrying out the swabbing. When this was completed, the man's slip and tube were handed to the person responsible for writing the name and code number on it with a wax pencil.

Swabbing Procedure

The throat was carefully swabbed, with particular attention being given to the tonsils. This swab was then transferred to the sterile tube. The other swab was inserted first into one nostril and then into the other with a rotating motion, to make sure it carried well back into the nasal passage. The tube was then flamed and both swabs tightly plugged into it.

The first 2,000 swabs had the ends of the applicator sticks broken off just below the rim of the tube. The hope was that the plug could be more tightly inserted into the tube and thus reduce possible contamination. The laboratory, however, informed us that it would be better if the applicators were not broken. In view of this, the wooden applicator ends of the remaining swabs were allowed to protrude from the tube.

As the survey progressed, it was found that it required approximately half a minute per man to carry out the swabbing and half a minute to fill out the slip. The greatest number of persons surveyed in any one day was 386.

Each day, after all swabs had been taken, the names and code numbers on the tubes were checked against the list. The tubes were packed in cotton and forwarded to Toronto, by air express, along with a copy of the list. Specimens done by the Department of Veterans Affairs Hospital, Victoria, were delivered by messenger.

The effort was made to have the week's quota of swabs from 500 persons reach the Ontario Laboratories by Wednesday of that week, if possible. However, as the numbers to be swabbed became fewer during the third week, it was necessary for the unit to move frequently, as only small numbers of persons were available for examination at any one place. In view of this delay, it was found necessary to ship by noon of the same day the swabs taken in the morning; those taken in the afternoon were shipped the following morning.

As the survey progressed, it became apparent that it would be almost impossible to obtain the fourth week's quota of 500 persons from naval personnel. It was decided to request the co-operation of army personnel stationed at Gordon Head Barracks and Work Point Barracks, B.C. The commanding officers of the two artillery regiments agreed to have the survey made, and in two days' time swabs from 500 persons were obtained. The procedure outlined above was followed for taking and shipping the swabs.

Laboratory Procedures

A total of 2,093 specimens were taken. The Ontario Department of Health laboratories in Toronto examined 1,996 specimens, while the remaining 97 examinations were done at the D.V.A. hospital laboratories in Victoria.

Ontario Department of Health Laboratories

In Toronto all specimens were examined to determine the presence or absence of *C. diphtheriae* only. Of the 1,996 specimens received, 1,644 were inoculated on Loeffler's medium and incubated for 24 hours, after which smears were made from the surface growth. One hundred and seventeen (7.1%) showed morphologically suspicious organisms which required further examination. These cultures were sub-cultured on chocolate-tellurite (McLeod) medium,[5] incubated for 48 hours, and also plated on serum-tellurite medium.[6] From these the suspicious or typical colonies were picked.

Twelve cultures isolated were sufficiently suggestive to warrant doing fermentation reactions (dextrose, saccharose, dextrin) and virulence testing.

The remaining 352 specimens were inoculated directly on chocolate-tellurite medium without culture on Loeffler's. Of these, 27 (7.6%) showed organisms which required further work, but none proved to be *C. diphtheriae*.

Department of Veterans Affairs Hospital Laboratory

The specimens delivered to the D.V.A. hospital, Victoria, were cultured within two hours of swabbing. The swabs were placed on Loeffler's serum slopes, Hoyle's tellurite-laked horse-blood plates,* and plain. Smears were made following 24-hour incubation at 37°C., and any colonies on the tellurite plates were transplanted to Loeffler's serum slopes for recheck. All 2,093 specimens were reported negative for *Corynebacterium diphtheriae*.

Discussion

This study was carried out as part of a survey which is being conducted in various parts of Canada in an attempt to determine the diphtheria-carrier rate in the country. Periodic surveys of this type would indicate any variation.

Although the procedures followed are generally adequate for the detection of cases of diphtheria, the occasional carrier in whom the organisms might be present in only small numbers might have been missed. Circumstances did not permit a more complete bacteriological examination, but even with this limitation it is interesting to note that no carriers were found, despite the fact that 10 persons (0.5%) claimed to have been diphtheria carriers sometime previously. Dr. Donald T. Fraser of the School of Hygiene and Connaught Medical Research Laboratories, University of Toronto, has indicated that these results are not out of harmony with those obtained elsewhere in Canada.

The failure to detect carriers should not be a deterrent to an active program of immunization. The only sure defence against an epidemic of diphtheria is a high level of immunity, which in the absence of recurrent outbreaks can be achieved only through artificial immunization.

It should be emphasized that this group was not representative of the general population, in that all those swabbed had received toxoid and may have received booster doses. Furthermore, the age range was from 18 to 45 years and the great majority were men.

The Department of Veterans Affairs hospital laboratory in Victoria, when carrying out the examination for *C. diphtheriae*, found that cultures grown on blood agar plates showed a fairly high percentage of this group to be carriers of haemolytic streptococcus, group A.

The Ontario Department of Health laboratories routinely plate the specimens on blood agar, but were not able to do this with such a large number of additional specimens.

*5% blood in Difco blood agar base.

SUMMARY

Nose and throat swabs from a group of 2,093 persons from the naval base at Esquimalt, British Columbia, and the army barracks at Gordon Head and Work Point, British Columbia, were examined bacteriologically for the presence of *C. diphtheriae*. No carriers were found.

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The Consumption of Recommended Foods by Children in Relation to Sex, the Use of Sweet Foods, and Employment of Mothers

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IN recent years there have been a number of studies on the use of foods by school children in various parts of Canada. These have shown that considerable percentages of children do not consume foods in the quantities recommended in Canada's Food Rules. The present study is similar in method and in general observations. However, there was an opportunity to investigate the effect of liberal use of sweet foods on the consumption of other foods, to ascertain whether food use differed for boys and for girls, and to consider the influence of regular out-of-home employment of mothers on the nutritional quality of meals obtained by children.

Study Group and Methods

Pupils in grades 6, 7 and 8 were selected as subjects because previous observations indicated that at ages 11, 12 and 13 children tend to adopt food habits which they regard as adult. Subjects were pupils in two schools in a suburban area of Greater Toronto. Racial origin was largely Anglo-Saxon and the families could be described as middle-class, living in individual houses.

Through the cooperation of principals, teachers requested pupils to record complete food intakes for seven days, using small booklets which were provided. The period of recording was April 14-20 inclusive, 1953. Stress was laid on the inclusion of all foods and beverages with the exception of water. Homy measures of quantities were requested. Apparently complete records were obtained from 242 girls and 202 boys.

For each record, total intakes of all foods for the week were computed and each total divided by seven to obtain the average daily consumption. Average figures were evaluated for adequacy in terms of Canada's Food Rules. For all foods listed in these rules, an intake of one-half or less of the recommended quantity was rated as "unsatisfactory". In the case of vitamin D, a record of daily use of any preparation likely to supply 400 units was considered satisfactory. Cake, pastry, candy, sugar and soft drinks were classified as sweet foods and the average daily consumption of three or more servings was rated unsatisfactory.

Results

The percentage of children having unsatisfactory intakes of various foods and of vitamin D are shown in Table I. The situation regarding food avail-

ability and price at the time of the study should be noted. Bottled milk was readily obtainable by house delivery or retail purchase at a price which had been constant for at least one year. Oranges and grapefruit were in excellent supply at a remarkably low price. Domestic vegetables, other than potatoes, were not in good supply but imported vegetables were available at a fairly reasonable price. The price of meat had declined to some extent before the study.

The percentage of children having a low milk intake was less than had been anticipated. The situation with regard to citrus fruit and vegetables is obvious. The most expensive food, meat, was used in liberal amounts. Inspection of the food records shows a general lack of interest in breakfast. It is interesting to compare the results of the present study with those obtained from a similar group of children in an adjacent area in 1948.[1] In the 1948 study more children obtained satisfactory supplies of milk, citrus fruit and vitamin D, but fewer ate recommended amounts of meat. The cost of citrus fruit was about the same in 1948 and in 1953 but the prices of milk and of meat, especially, were markedly increased in 1953.

It was suggested that girls aged 11-13 might have more unsatisfactory food intakes than boys in this age group. Table I gives the percentages of girls and boys rated as having unsatisfactory intakes. The differences between the sexes are not sufficiently great to be significant.

TABLE I

Proportion of Children with Unsatisfactory Intake of Various Foods

Food	444 Children, both sexes	242 Girls	202 Boys
Milk	25	24	26
Citrus fruit	40	42	37
Vegetables	50	46	54
Cereals	74	79	69
Meat	5	2	9
Vitamin D	90	86	95
Sweets	16	18	13

In the past three years there has been considerable educational emphasis in the Toronto area on the use of sweet foods by children. A restriction in the consumption of sweet foods has been urged for three reasons: evidence that a liberal use of sweet foods promotes dental caries; the assumption that sweet foods dull the appetite and may lessen the consumption of other foods; and the relatively poor return in nutritive value for the money spent on such foods. In the present study an arbitrary division of children was made on the basis of the use of sweet foods. Three or more servings a day was considered unsatisfactory, while less than three servings was rated satisfactory. This classification is, of course, open to question. However, this rating was used

[1]Shaver, E. M., Esler, E. M., Mosley, W., and McHenry, E. W., Canad. J. Pub. Health, 1948, 39:395.

in the preparation of Table II, which shows percentages of children having unsatisfactory supplies of other foods.

TABLE II

Comparison of Food Intake of Children Having a Liberal Use of Sweets With Those Having a Sparing Use of Sweets

Food	Percent children having unsatisfactory intakes	
	70 Children—Liberal use of sweets	374 Children—Sparing use of sweets
Milk	27	24
Citrus fruit	29	42
Vegetables	43	51
Cereals	80	73
Meat	0	6
Vitmain D	90	90

In interpreting the data in Table II it should be noted that the number of children classified as using liberal amounts of sweet foods is small and that the basis of classification is arbitrary. However, the information does not support a contention that liberal use of sweet foods decreases the intake of other foods. In fact, a greater proportion of the children rated as liberal users of sweet foods had satisfactory consumption of citrus fruit and vegetables.

During the period of the study it became apparent that a considerable number of children had mothers who were regularly employed outside the home. This provided an opportunity to compare the food intake of these children with those whose mothers were at home. The results are shown

TABLE III

Comparison of Unsatisfactory Food Intake of Children Whose Mothers are Employed Outside the Home With Those Whose Mothers are at Home

Food	Percent children having unsatisfactory intakes	
	111 Children with mothers employed	333 Children with mothers at home
Milk	27	24
Citrus fruit	42	39
Vegetables	51	49
Cereals	76	73
Meat	7	4
Vitamin D	96	87
Sweets	17	15

in Table III. No significant difference in food use between the two groups of children is apparent. It does not seem that the absence of the mother from the home each working day made worse an already unsatisfactory picture of food use.

Summary

1. A study of food use for one week of 444 urban children aged 10-14 years showed that large percentages had less than half the recommended amounts of milk, citrus fruit, vegetables, whole grain cereals and vitamin D. Nearly all had sufficient meat.
2. No significant difference in food intake was evident between girls and boys.
3. Using arbitrary rating of the intake of sweet foods, a greater percentage of liberal users of sweet foods had satisfactory supplies of citrus fruit and vegetables. The reported data do not support a contention that liberal use of sweet foods lessens the intake of other foods.
4. The percentage of children with unsatisfactory supplies of recommended foods was not increased appreciably by the regular out-of-home employment of the mothers.

It is a pleasure to acknowledge indebtedness to Dr. William Mosley, Medical Officer of Health, and to the school principals and teachers whose cooperation made the study possible.

Salmonella Typhimurium Infection in 1952 Turkey Flocks - A Public Health Hazard

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Regina

EVERY year the organism *Salmonella typhimurium* (synonym *Aertrycki*) is convicted of having provoked more outbreaks of Salmonella food poisoning than any other member of this large genus. Feig,[3] in a report to the United States Public Health Service, states that twenty-five per cent of Salmonella outbreaks are due to typhimurium. Fortunately most of these infections are of a mild nature, but occasionally serious cases do occur, so that this pathogen perhaps deserves more respect than is commonly tendered. The human carrier state for this organism is often difficult to clear up, perhaps due to repeated re-infection, and so the hazard is magnified and perpetuated.

The principal reservoirs of such organisms are man and the birds and animals with which he comes in contact. The carrier state is common to all, but poultry seem to have the added faculty of transmitting the organism through the egg. In Saskatchewan, a large proportion of the turkey poults hatched from imported eggs were found to be infected in 1952. Mortality and morbidity were extremely high in many of the infected flocks. The morbidity was not limited to the poults, but involved the handlers of at least one and possibly several flocks. The majority of the infected birds recovered but continued to excrete *S. typhimurium* in their droppings for at least thirty days after apparent recovery. There is good reason to believe such birds will reach market in an apparently healthy condition. However, because of the practice of marketing turkeys in a semi-dressed state, they may be capable of infecting the cooks or other handlers who prepare them for the table.

This report is concerned with relating the association of *S. typhimurium* in turkey poults and gastroenteritis in their handlers, with the objective of drawing attention to what may be an increasing hazard.

The first recorded outbreak in Saskatchewan occurred near North Battleford and involved all members of a family of seven. The mother became so

[1]Animal Pathologist.
[2]Director.
[3]Feig, Milton: Diarrhoea, Dysentery, Food Poisoning and Gastroenteritis, Am. J. Pub. Health, 1950, *40*:1372.

ill that she sought medical attention and was hospitalized as a suspected typhoid-paratyphoid fever case. Stool specimens from the patient and all other members of the family were referred by the medical health officer,* and each yielded S. *typhimurium* by culture. The medical health officer investigated and the only outside contact recognized was the receipt of twenty-five turkey poults from a commercial hatchery a few days before the date of the outbreak. Twenty-three of these had developed a whitish diarrhoea and died within a week. One of the survivors and one of the dead birds were taken for examination. Faecal samples of each yielded S. *typhimurium*. The live poult appeared to be in good health and was maintained in the laboratory for thirty days. During this time, S. *typhimurium* was consistently recovered from the droppings.

It was desirable to learn whether the poults had infected their handlers, or vice versa. An investigation was made through the Federal Poultry Inspector as to the fate of other poults of the same hatch shipped to other points from the same hatchery.

The inspector had before him a report of extremely high mortality in a flock of three thousand poults set out at Melville, over one thousand of these poults having died in the first ten days. A number of surviving but droopy birds were referred for examination. S. *typhimurium* was recovered from each of these birds. It was recommended that the birds be treated with sodium sulfamethazine, and with this therapy the losses reportedly tapered off.

The second human outbreak was suspected on the same premises. The rancher's wife and daughter described an outbreak of intestinal "flu" shortly after receiving the turkey poults. They had helped "dip" the poults and possibly had contracted the infection in the process. Their illness was acute but did not warrant medical attention. No stool cultures were attempted on the patients, since a month had elapsed between the onset of their symptoms and the field investigation.

The third outbreak was suspected when a physician referred a blood specimen to the Provincial Laboratories with a request for culture. The patient, the wife of a butcher-locker plant operator, was seriously ill with fever and diarrhoea. S. *typhimurium* was recovered from this specimen. An effort to locate the source of the organism was unsuccessful. Repeated stool specimens of the husband and his assistant were consistently negative. The woman had no other outside contacts, but it was suspected that some phase of meat-handling operations had brought home the organism. Supporting this suspicion was the fact that her husband had removed and dressed some frozen turkeys a short while before his wife took ill.

Further isolations of S. *typhimurium* from other flocks have since been made. In all cases, either poults or the egg were purchased from the same distributor in British Columbia, who had in turn imported them from the southern United States. S. *typhimurium* was readily detected in one flock of eighteen thousand birds over two months of age. There is a real hazard that such birds will carry

*Dr. C. F. W. Hames, Medical Health Officer for the North Battleford Health Region.

these organisms to their ultimate purchaser, who will dress the birds for his own table.

Summary

1. There is an apparent increase in mortality of turkey flocks due to *S. typhimurium*.

2. Several human cases from handling infected birds are reported.

Conclusion

In the light of such findings it seems safe to predict that *S. typhimurium* will continue to rank as the number one offender of Salmonella organisms provoking food poisoning, and may very well increase its lead.

Canadian Journal of Public Health

THE CANADIAN PUBLIC HEALTH ASSOCIATION

THE FORTHCOMING forty-first annual meeting of the Canadian Public Health Association, which will be held in Toronto on October 1 and 2, will present important recommendations from the Executive Council concerning membership and the establishing of adequate financial support.

Four years ago the Council approved a plan of establishing provincial public health associations, functioning as provincial branches of the national organization and holding annual meetings. Since then, the following branches have been organized: la Société d'Hygiène et de Médecine Préventive de la Province de Québec, the Ontario Public Health Association, the Alberta Public Health Association, the Atlantic Branch (serving Nova Scotia), the Manitoba Public Health Association, the New Brunswick-Prince Edward Island Association, and the British Columbia Branch. In April of this year, at the sessions of the Public Health Institute convened by the Provincial Department of Health, the formation of a British Columbia Branch was recommended by Dr. G. F. Amyot, Deputy Minister of Health, Dr. G. R. F. Elliot, Assistant Deputy Minister, and Dr. Stewart Murray, Senior Medical Health Officer of Vancouver. The Public Health Institute has been an essential part of the Provincial public health service. The provincial association will have an effective membership which has already expressed its keen interest in the national work of the Canadian Public Health Association. In Saskatchewan, the remaining province to be organized, it is expected that the Health Officials' Association will be reorganized as the Saskatchewan Public Health Association.

The advantages of providing, in the functioning of the Canadian Public Health Association, for the establishing of provincial branches are at once evident when it is remembered that the interest of the public health workers in the ten provinces cannot be maintained through the holding of one national meeting, which of necessity must be convened in widely distant centres from year to year. This plan of provincial associations, each holding an annual meeting, has therefore marked a forward step in the development of the Association. The national meeting will be held in conjunction with meetings

of the provincial branches, thus bringing to each in turn the sessions of the Executive Council and the added stimulus of the national conference.

Each of the provincial associations is holding a meeting this year. In May the Quebec Branch, la Société d'Hygiène et de Médecine Préventive de la Province de Québec, held a regional conference in Sherbrooke, and in September the Société will hold its general meeting in Quebec City with the Association of French-speaking Physicians of North America. The Atlantic Branch held its third annual meeting in Halifax on June 15 and 16, under the chairmanship of Dr. J. E. LeBlanc. One hundred and fifteen were present from all parts of Nova Scotia. The program was particularly well planned, and if the standard is maintained, the meeting will be quickly recognized as constituting a refresher course on immediate problems.

Four meetings are planned for the fall. The Alberta Public Health Association will meet in Edmonton on September 3 and 4. In October there will be three meetings: the forty-first annual national meeting in Toronto on October 1 and 2, in conjunction with the annual meeting of the Ontario Public Health Association; the meeting of the Manitoba Public Health Association in Winnipeg on October 15 and 16; and the meeting of the New Brunswick-Prince Edward Island Association in Fredericton, on October 20.

Thus it can be seen that excellent progress has been made in the organization of provincial branches and that the plan adopted four years ago has placed the Association in a most promising position.

In has been appreciated by every public health worker that the Canadian Public Health Association renders valuable services, but it has not been possible in the past to bring the Association into vital contact with its members. It is the professional society of those who are engaged in public health work. However, it lacks the cohesion of common professional backgrounds that constitute the strength of medical, dental, nursing and other professional societies. Individual members continue to be much more conscious of their participation in the society of their basic professional training than they are of membership in the Canadian Public Health Association. The organization of provincial public health associations will help to overcome this handicap.

The Canadian Public Health Association has during its forty-odd years served as a means of providing instruction and supplying information. It has been so regarded by the Federal Department and the Provincial Departments of Health. The files of the Canadian Journal of Public Health constitute the only systematic record of public health progress. To find the origin of any given program, one turns to its pages. The Journal has been supplied through the years to the leading libraries in government departments, universities, and institutions. All of the work that has been contributed in the maintenance of the Association is justified by the contribution of the Journal alone. The Association can be proud of this accomplishment.

With only three exceptions, in war years, the Association has held a national meeting every year since 1912, attended by the leading public health authorities. Here again, the Association has influenced public health thinking, government action, and public interest. Through these meetings, resolutions

embodying the views of Canadian public health authorities have been endorsed. The Canadian Public Health Association speaks for the public health profession.

The Association is rendering much of its service through the work of committees. The Committee on Professional Education is of special importance. Through it the qualifications for various public health appointments have been established. In collaboration with the Committee on the Certification of Sanitary Inspectors, it has provided a correspondence course which constituted the basis of training for sanitary inspectors from 1942 until the provision of courses in the University of Montreal, the Manitoba School for Sanitary Inspectors, and the Department of Health of Ontario. More than eight hundred men have qualified for the Certificate in Sanitary Inspection (Canada), granted by the Association.

Through other committees the Association has made important contributions in the field of vital statistics, maternal and child hygiene, and the extension of public health laboratory work. Through its Committee on Social Security the Association is studying the place of public health and preventive medicine in a national health program. Of primary importance to departments of health, as well as to individual health workers, has been the interest of the Association in reviewing salaries and in recommending minimum salary schedules for the various types of public health appointments.

As stated before, the Executive Council this year will consider the interrelationship of membership between the provincial organizations and the national association and the ways in which the essential financial support for the national association may be provided. In regard to the latter, the Provincial and Federal Departments of Health have acceded to the Association's request for the recognition of its work on the basis of services rendered. This action is evidence of the appreciation by these authorities of the work of the Association in the advancement of public health in Canada.

Encouraging as this support is, the maintenance of the national association presents a serious problem in view of the continual increases in the cost of publishing the Journal and in financing the annual meetings. The situation calls for the most careful consideration and the adoption of plans which will assure the successful development of the provincial associations and the maintenance of the national association. The Canadian Public Health Association is credited with soundness of judgment and its recommendations have been welcomed by Federal, Provincial, and municipal health authorities. The national meeting this year offers a most attractive program. It reflects the combined efforts of the Ontario Public Health Association and the national organization. Adequate representation from all the Provinces is needed if the Association is to go forward and make the contribution that it should to Canadian public health.

CANADIAN PUBLIC HEALTH ASSOCIATION
Forty-First Annual Meeting
ONTARIO PUBLIC HEALTH ASSOCIATION
Fourth Annual Meeting
Royal York Hotel, TORONTO
October 1 and 2, 1953
(Executive Meetings September 30)

DIRECTORY OF SESSIONS

Wednesday, September 30

2.15 p.m.—*Executive Council*, Canadian Public Health Association. Private Dining Room No. 7.
—*Executive*, Ontario Public Health Association. Private Dining Room No. 6.

Thursday, October 1

8.30 a.m.—*Registration*. Convention Foyer.
9.30 a.m.—*Section Meeting*: *Public Health Nursing*. Library.
10.00 a.m.—*Minister's Conference*. Concert Hall.
—*Section Meetings*:
Sanitation. Private Dining Room No. 10.
Vital & Health Statistics and *Epidemiology*. Private Dining Room No. 8.
12.15 p.m.—*Luncheon*. Tudor Room.
2.15 p.m.—*Section Meetings*:
Health Officers, *Public Health Nursing*, and *Dental Public Health*. Concert Hall.
Public Health Education. Private Dining Room No. 10.
Veterinary Public Health. Library.
7.00 p.m.—*Annual Dinner*. Ballroom. Speaker: Dr. HARRY M. WEAVER, Director of Research, The National Foundation for Infantile Paralysis, New York.

Friday, October 2

9.15 a.m.—*General Session*. Ballroom.
12.15 p.m.—*Luncheon*. Parlour A.
2.15 p.m.—*Section Meetings*:
Health Officers and *Sanitation*. Ballroom.
Public Health Education and *Public Health Nursing*. Library.
Dental Public Health. Private Dining Room No. 10.
Vital and Health Statistics. Private Dining Room No. 8.

Saturday, October 3

TRIP FOR OUT-OF-TOWN MEMBERS

NEWS

British Columbia

AT THE ANNUAL Public Health Institute, held in Vancouver in April, a meeting was held to discuss the formation of a British Columbia Branch of the Canadian Public Health Association. At this meeting the establishment of a branch was endorsed and an executive elected. Application for Branch membership has duly gone forward to the executive of the Canadian Public Health Association.

DURING APRIL Vancouver enjoyed a visit from Sir Allen Daley, formerly the Chief Medical Officer for the London County Council. Sir Allen is returning to England after spending some time in Australia under the Nuffield Foundation. While he was in the city, such groups as the Medical Association, the Metropolitan Health Services, and the Faculty of Medicine at the University of British Columbia gained a great deal from Sir Allen's informal talks.

MISS ALICE MILLS, director of women's programs with the National Safety Council, Chicago, spoke to Vancouver's Traffic and Safety Council in April. Some senior members of the Vancouver Health Department met Miss Mills to discuss plans for the formation of a home-safety program within a health department.

BRITISH COLUMBIA sanitary inspectors will welcome their fellow-workers from across Canada at the annual meeting of the Canadian Institute of Sanitary Inspectors, which will be held in Vancouver August 24, 25 and 26.

THE SIXTEENTH of eighteen proposed health units in this Province commenced full-time operation in June. Known as the Selkirk Health Unit, this unit, with headquarters in Nelson and sub-offices in New Denver and Nakusp, will serve school districts six, seven, eight and ten. Dr. H. T. Lowe, who received the Diploma in Public Health from the University of Toronto this year, has been appointed director.

MRS. LORRAINE ARSENAULT, nutrition consultant with the Department of Health and Welfare, now maintains her office at the Health Branch, 2670 Laurel Street, Vancouver. Mrs. Arsenault will serve in a consultant capacity to various institutions throughout the Province.

MRS. KAY BEARD, consultant in public health education, has resigned from the Department. Mr. R. H. Goodacre, who received the Certificate in Public Health from the University of Toronto last year, has succeeded Mrs. Beard.

DR. A. F. BALKANY, formerly director of the Peace River Health Unit, has returned from the University of Toronto, where he received the Diploma in Public Health. Dr. Balkany is now director of the West Kootenay Health Unit.

DR. D. L. CLARKE, who spent last year at the Harvard School of Public Health, has returned to British Columbia and will take over as director of the South Okanagan Health Unit on the departure of Dr. D. M. Black for post-graduate training.

Saskatchewan

THE DEPARTMENT of Public Health's newly formed Division of Physical Restoration is now administering crippled children's services, formerly under the Division of Child Health, and has expanded its program to include the physical restoration of adults as well, particularly in the field of post-poliomyelitis. Directing this division is Mr. Keith S. Armstrong, M.A., D.S.W., formerly assistant director of child health in charge of crippled children's services and executive director of the Saskatchewan Council for Crippled Children and Adults. In order to make the Department's physical restoration program province-wide, it has taken over the administration of the Saskatoon Rehabilitation Centre operated previously by the Council. It has also extended treatment facilities in Regina. Due to limited space in the Regina General Hospital for the Cerebral Palsy Centre, the south wing of the Saskatchewan Boys' School has been opened for treatment services. Additional physical therapists have been employed and classroom facilities have been expanded.

APPROXIMATELY 140 delegates attended the 17th annual meeting and first national conference of the Canadian Council for Crippled Children, Inc., which was held at the Hotel Saskatchewan in Regina from June 17 to 19. A very successful panel on cerebral palsy was conducted under the leadership of Dr. A. E. Buckwold, medical director of the Saskatoon Rehabilitation Centre, and Dr. Fred Day, director of cerebral palsy in Alberta. Other members of the panel were Dr. A. Stephens, director

of the McNeill Clinic in Saskatoon; Dr. S. C. Best, director of child health, Regina; Keith S. Armstrong, director of physical restoration, Regina; and Mrs. H. M. Schneider of Saskatoon, representing parents.

THE EIGHTH WESTERN CANADA INSTITUTE for Hospital Administrators and Trustees was held at the University of Saskatchewan, Saskatoon, from June 15 to 19. Arrangements for the Institute this year were largely the responsibility of the Saskatchewan Hospital Association through its executive secretary, Mr. E. V. Walshaw. The coordinating committee chairman was Dr. Angus C. McGugan, D.P.H., M.A.C.H.A., superintendent of the University of Alberta Hospital, Edmonton. Several representatives from the Department of Public Health were in attendance and addresses were given by the Hon. T. J. Bentley, minister of public health, and Dr. F. B. Roth, deputy minister. Problems relating to hospital administration which are of concern to boards of trustees and administrative staff were the essential items under consideration by the assembly.

Manitoba

DR. JEAN R. DUPONT has been appointed medical director of the Virden Local Health Unit, at Virden. She is a graduate of St. Boniface College (University of Manitoba) and Laval University, Quebec.

MISS MARGARET MUNRO, B.Sc.(H.Ec.), has been appointed to the position of junior nutritionist with the Bureau of Health and Welfare Education, Department of Health and Public Welfare. She succeeds Mrs. Carolyn Soderman, who is now living in Toronto.

A SURVEY OF PUBLIC HEALTH NURSING SERVICES within the Department of Health and Public Welfare was recently completed by Miss Ruth B. Freeman, director of the Department of Public Health Nursing and associate professor of public health administration at the Johns Hopkins School of Hygiene and Public Health, Baltimore.

DR. W. McD. HAMMOND, visiting speaker at the annual meeting of the Canadian Medical Association, held in Winnipeg in June, addressed a meeting of the Section of Preventive Medicine, Department of Health and Public Welfare. Dr. Hammond also met with health department members to discuss the possible use of gamma globulin in the prevention of poliomyelitis.

Ontario

ELEVEN STUDENTS in the sanitary inspectors' training course, currently being held under the auspices of the Ontario Department of Health at the Ryerson Institute of Technology, Toronto, will receive field training in ten health departments and health units throughout the Province during July and August. The men will acquire practical experience, under the supervision of qualified health personnel, in environmental and food sanitation, as well as other phases of health work. They will be assigned to health departments in Ottawa, Peterborough, Scarborough and Etobicoke, and the following health units: Kenora-Keewatin-Dryden Area, Peel County, Northumberland, York County, Fort William and District, and East York-Leaside.

Upon completion of the course, which consists of theory and practical work, students passing the examinations conducted by the Canadian Public Health Association will receive the Certificate in Sanitary Inspection (Canada) and may be employed in any of the Province's health departments or units.

New Brunswick

DR. R. J. DOLAN, director of hospital services, represented the Department of Health and Social Services of the Province at the meeting of the Continuing Committee on Hospital Statistics held at the Dominion Bureau of Statistics on May 13th to 15th. He also attended the meeting of the Canadian Hospital Association in Ottawa on May 18th to 20th.

AN EXECUTIVE MEETING of the New Brunswick-Prince Edward Island Branch of the Canadian Public Health Association was held in Fredericton on June 8. Those present included the president, Dr. J. A. Melanson, and the 1st vice-president, Dr. O. H. Curtis. The revised constitution and by-laws of the N.B.-P.E.I. Branch were discussed and are to be brought forth at the next annual meeting, which will be held in Fredericton on October 20th.

EARLY IN JUNE the Grand Council of the Canadian Cancer Society held their annual meeting in Fredericton. The three-day conference was under the chairmanship of the president of the Grand Council, Mr. F. G. Butterfield, of Regina. On the last day of the meeting, June 9th, the Government of the Province was host to the delegates at a banquet presided over by the Honourable W. J. West, the acting premier. The delegates and guests were addressed by the Honourable J. F. McInerney, M.D., Minister of Health and Social Services.

THE ANNUAL MEETING of the Maritime Hospital Association was held at the Algonquin Hotel, St. Andrews, on June 10 to 12. The session was attended by the Honour-

able J. F. McInerney, M.D., Minister of Health and Social Services; Dr. J. A. Melanson, Chief Medical Officer; Dr. R. J. Dolan, Director of Hospital Services; and Mr. L. M. Byron, departmental accountant of the Department of Health and Social Services.

THE FOURTH BIENNIAL MEETING of the Maritime Conference on Social Work, held in Fredericton June 16 to 18, attracted more than 150 social workers and persons interested in social-welfare development of the four Maritime Provinces. The Honorary President, the Honourable J. F. McInerney, M.D., Minister of Health and Social Services, Fredericton, presided at the opening sessions, at which the guest speakers were the Honourable H. L. Pottle, M.D., Minister of Public Welfare for Newfoundland, and Dr. Arthur J. Altmeyer, former Commissioner for Social Security, Washington. The officers for the conference were Mr. R. H. Scott, Provincial Child Welfare Officer, president; Mr. W. J. Whittingham, treasurer; and Miss Kathleen C. Morrissy, secretary, all of Fredericton. The new officers elected are Mr. L. T. Hancock, Miss Pauline MacDonald, and Mr. George Hart, of Halifax. The next conference will be held in Nova Scotia in 1955.

A SIX-DAY COURSE for waitresses, sponsored by the Fredericton Junior Chamber of Commerce in cooperation with the New Brunswick Travel Bureau, was attended by more than thirty employees of local restaurants. The District Medical Health Officer, Dr. J. R. Allanach; the Provincial Sanitary Engineer, Mr. A. J. Cameron; and the Senior Nutritionist, Miss Florence Swan, from the Department of Health, lectured and led group discussions, as well as participating in other activities of the course.

DURING THE LATTER PART of June the Canadian Public Health Association, in co-operation with the New Brunswick Department of Health, held the examinations for the Certificate in Sanitary Inspection (Canada) in Fredericton for the first group of sanitary inspectors employed by Boards of Health in the Province, under assistance from the Federal Health Grants. Two candidates presented themselves for examination before an examining board headed by Dr. J. R. Allanach, District Medical Health Officer, who acted as chairman, assisted by Mr. A. J. Cameron, Provincial Sanitary Engineer, and Mr. George Hamilton, C.S.I.(C.), of Saint John. While only two inspectors took the correspondence course and the examinations this year, it is expected that the remaining eight now employed by the local Boards of Health will enroll for the 1953-54 course in October.

Nova Scotia

THE THIRD ANNUAL MEETING of the Atlantic Branch of the Canadian Public Health Association was held in the auditorium of the Victoria General Hospital in Halifax on June 15th and 16th. Over 115 members were present. The first general session was presided over by the president, Dr. J. E. LeBlanc of Pubnico. Papers were presented on fluoridation by W. G. Dawson, D.D.S., and on restaurant control by W. J. Chisholm, C.S.I.(C.), sanitary inspector. The guest speaker was Dr. R. D. Defries, D.P.H., director of the School of Hygiene and Connaught Medical Research Laboratories, University of Toronto, who discussed recent advances in virus research. The afternoon was given over to a round-table discussion of a practical program of prenatal and infant care which was chaired by Dr. H. B. Atlee, head of the Department of Obstetrics and Gynaecology of Dalhousie University.

At the annual banquet the speakers were the Honourable Harold Connolly, Minister of Public Health and Welfare, Mayor Richard A. Donahoe, and Miss Electa MacLennan, director of the School of Nursing of Dalhousie University.

The second morning was devoted to a paper on home care nursing by Miss Jean Forbes, R.N., district superintendent of the Victorian Order of Nurses, followed by a round-table discussion on modern methods of control of the chronic diseases, with special emphasis on diabetes, rheumatism and cardiovascular disease. This was chaired by Dr. C. B. Stewart, professor of epidemiology, Dalhousie University.

The afternoon session included a paper on "A Preventive Medical Program in a General Hospital" by Dr. M. R. MacDonald, Assistant Superintendent, Victoria General Hospital, Halifax. This was followed by a general business session. The officers elected for the ensuing year were: President, Dr. H. E. Kelley, Middleton; 1st Vice-president, Miss P. J. Lyttle, R.N., Halifax; 2nd Vice-president, Dr. A. C. Guthro, Little Bras d'Or; Secretary-treasurer, Dr. J. E. Hiltz, Kentville; Executive, Miss Jean Forbes, R.N., Halifax, Miss Electa MacLennan, Halifax, Dr. G. G. Simms, Halifax, Mr. W. J. Chisholm, Sydney, and Dr. C. E. A. DeWitt, Wolfville.